dioxide with the blood and tissues occurs. The fundamental abnormality in asthma is inflammation of the inner lining of the airways with swelling, secretion and narrowing of the lumen and air passage. There is also contraction of the bronchial smooth muscle surrounding the airways with resultant further narrowing of the lumen.

The principles of management of asthma involve firstly the prevention and treatment of airway inflammation and secondly relaxation of the bronchial smooth muscle.

Detailed analysis of asthma deaths has revealed that many result from lack of the knowledge about the condition on the part of many patients, and inadequate treatment strategies. The National Asthma Campaign was formed with the aims of reducing the mortality and morbidity in asthma, maintaining normal airway function and a normal lifestyle for patients with asthma. Constituent bodies are respiratory physicians (Thoracic Society of Australia and New Zealand), family doctors (Royal Australian College of General Practitioners), pharmacists (Pharmaceutical Society of Australia) and the Asthma Foundations of Australia.

The aims of asthma management are to minimise symptoms, to maximise lung function and maintain function at its best at all times, identify trigger factors and prevent the development of permanently abnormal lung function, to reduce the risk of death from an acute attack and to achieve the best quality of life for the person with asthma.

The Six Step Asthma Management Plan outlines these principles.

1. *Assess asthma severity*

Assess lung function when the condition is stable, not during an acute attack.

2. *Achieve best lung function*

Treat with intensive asthma therapy until the 'best' lung function is achieved as shown by measurement of spirometry or PEF.

3. *Maintain best lung function—avoid trigger factors*

Identify and avoid trigger factors and inappropriate medications. Peak flow monitoring can be helpful here.

4. *Maintain best lung function with optimal medication*

Treat with the least number of medications and use the minimum doses necessary.

Ensure the patient understands the difference between 'preventer' medications which combat inflammation and 'reliever' which relax bronchial smooth muscle.

Take active steps to reduce the risk of adverse effects.

5. *Develop an action plan*

Discuss and write a plan for the management of exacerbations.

Detail the increases in medication doses and when and how to gain rapid access to medical care.

6. *Educate and review regularly*

Ensure patients and their families understand the disease, the rationale for their treatment and how to implement their action plan.

Emphasise the need for regular review, even when asthma is well controlled.

The concept of the individual asthmatic person taking an active role in the management of his or her condition as part of a team effort with their local doctor and pharmacist is very important in successful management. The best patient outcome is most likely to be achieved when there is a close working relationship between an interested doctor and an informed patient.

BRUCE KNOX

POLLEN AND THE WEATHER

Pollen grains occur in conifers and flowering plants and carry the male gametes. They are released into the air for fertilisation so that pollen is a natural component of the atmosphere in all seasons, but is especially numerous in spring and early summer in temperate climates. Susceptible humans, breathing the pollen-laden air, develop the symptoms of hay fever and asthma. These allergic diseases are triggered by the presence of certain proteins and glycoproteins in some kinds of pollen that act as allergens. These are environmental proteins or glycoproteins, which interact with the human immune system and trigger the allergic response. Given the dramatic increase in incidence of allergic disease that has occurred during the past 30 years, there has been increasing public concern about allergenic pollen. In this talk, we will examine when, where and in what quantities pollen allergens are present in the aerial environment and how it is affected by weather patterns.

Assessment of the amount of pollen in the air is obtained through use of spore traps, which provide quantitative incidence estimates per cubic metre of air sampled on an hourly to annual basis, as levels of efficiency depending on the size of the particle. A pollen calendar has been produced for Melbourne which shows this information graphically for 22 different types of pollen. Tree pollens predominate in winter: ash, birch, olive and wattle are the most allergenic in humans and, with the exception of wattle, are exotic horticultural species. In spring and early summer, grass pollen predominates (26% of the total pollen catch in Melbourne). Grass pollen is the major source of aeroallergens in the external environment, with about 60% of allergic humans being sensitised to this type of pollen. Weed pollens follow in summer, e.g. wall pellitory, which is now the dominant weedy plant in the CBD of Sydney and abundant in central Melbourne.

The seasonal total pollen counts vary widely from season to season, reflecting the intensity of flowering in any year. For Melbourne, the range is from 2319 to 8217 grains m^{-3} $year^{-1}$, similar to cities in Europe with temperate climates. The key factor regulating the seasonal appearance of grass pollen allergens is average temperature, which is precisely correlated with increasing grass pollen counts. This relationship can be used to predict the intensity of the pollen season. In Melbourne, wind direction is important, with northerly and northwesterly winds carrying high levels of grass pollen.

Rainfall during winter can be used as a predictor for the onset of the grass pollen season. Rain is negatively correlated, since the pollen is washed out of the air. Hay fever sufferers can breathe easy, but not so asthmatics. Rainfall merely changes the total allergen load in the atmosphere, causing the pollen allergens to disperse into the atmosphere aerosol. They exist in two different forms, allergen molecules and allergen-containing fine particles. As grass pollen grains remain living cells for only about one hour after entry into the atmosphere, most grains will be dead. In this condition, they are assumed to behave like a sponge in which allergen molecules diffuse from microchannels in the outer walls from the grains when moistened and become dispersed in the aerosol. Allergen-containing fine particles (starch granules) are produced when grains rupture osmotically during rainfall and are released from the single pore into the atmospheric aerosol. There are approximately 700 of these fine particles in each grain and their size ranges from 0.6 to 2.5 μm. In Melbourne on days following rainfall, a 50-fold increase in the number of particles m^{-3} air has been reported. Allergenic starch granules have been associated with epidemics of thunderstorm asthma during the grass pollen season.

A consequence of the release of allergen molecules into the aerosol is that these molecules are free to interact with other types of fine particles that are associated with air pollution. For example, diesel emitted carbon particles (DECP), each 60 nm in size but forming into fractal aggregates, originate from diesel engine exhaust emissions. Such fine particles have recently been shown to have serious health risks in several American cities with polluted air. These DECPs can become loaded with grass pollen allergen molecules, effectively concentrating the allergens, and suggesting a causal mechanism for the increased levels of asthma in cities during episodes of air pollution. Starch granules are, however, the only class of fine particles that have been tested clinically to show they are capable of eliciting an attack of asthma. There is now a need for quantification of the relative allergenic activities of these various fractions of grass pollen allergens and their contribution to the allergenic activity of the atmospheric aerosol.